

EXHIBIT C

DR. ARNOLD BRODY – JANUARY 2007 REPORT

DEVELOPMENT OF ASBESTOS-INDUCED LUNG DISEASES

ARNOLD R. BRODY, PH.D.
NORTH CAROLINA STATE UNIVERSITY

The diseases most commonly associated with exposure to asbestos fibers are:

- 1) Asbestosis: Scar tissue in the gas-exchanging regions of the lung;
- 2) Pleural Fibrosis or Plaques: Scar tissue formation in the pleural and sub-pleural connective tissue compartments of the lung;
- 3) Lung Cancer: Typically a malignant transformation of an epithelial cell type that lines a conducting airway;
- 4) Mesothelioma: Malignant transformation of pleural or peritoneal mesothelial cells.

The lung is comprised of several types of epithelial cells that line the airways and gas exchange areas of the lung. Epithelial cells make up the mucociliary escalator that protects the airways, and the Type I and II alveolar epithelium is the site of initial asbestos deposition and consequent injury at the level of the lung where gas exchange takes place. Mesenchymal cells underlie the epithelial components and produce the connective tissue matrix of the lung. Mesothelial cells line the outside of the pleural membrane (the visceral pleura) as well as the inner lining of the chest wall (the parietal pleura), and these cells line the peritoneal cavity. A variety of inflammatory and phagocytic cells respond to lung injury and mediate components of the developing disease process. Fibers of all types and all dimensions participate in the pathogenesis of the asbestos-induced diseases. These include the asbestos types chrysotile, crocidolite, amosite and tremolite. The sources of the fibers have been from mining or milling operations or from any occupational settings in which asbestos fibers are released. Significant exposures have also occurred in homes and in the environment as well as in buildings when asbestos-containing materials release fibers.

ASBESTOSIS and PLEURAL FIBROSIS:

Inhaled asbestos fibers deposit along all aspects of the respiratory tract. Fibers that land upon a normal mucociliary escalator will be moved to the mouth in a rapid clearance phase. Those fibers that pass through the airways and deposit on the alveolar surfaces can be dealt with in several ways: A) A proportion of the fibers will be cleared onto the mucociliary escalator by the natural movement of surfactant; B) A proportion of the fibers will be picked up by alveolar macrophages which carry the fibers to the escalator for clearance; C) A proportion of the fibers (approximately 20%) will be covered by the Type I alveolar epithelium and drawn into the cytoplasm of these cells for subsequent deposition in the underlying connective tissue (interstitial) compartment of the lung. Some proportion of these interstitial fibers will remain in the lung for the life of the individual while others are cleared slowly over time by interstitial macrophages and lymphatic flow. Shorter fibers are more likely to be cleared from the lung than longer

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fibers, and short chrysotile asbestos fibers have been found to accumulate in the pleural regions of human lungs.

Asbestos fibers which impact on the alveolar surfaces activate the 5th component of complement to produce C5a, a potent chemotactic factor for lung macrophages. This is the mechanism through which alveolar macrophages “find” the inhaled fibers for subsequent phagocytosis and are in a position to participate in the inflammatory and fibroproliferative process.

Macrophages, alveolar epithelial cells, and fibroblasts (the mesenchymal cell type that manufactures scar tissue) have been shown to phagocytize asbestos fibers. This induces the expression of a series of genes which code for a group of peptides known as “growth factors”. These factors control cell division (proliferation) and matrix (connective tissue) production, the two hallmarks of pulmonary fibrosis. When it is asbestos that stimulates the expression of the growth factors with consequent scar tissue formation, the result is asbestosis. Those fibers that are deposited in the pleural regions, either by inhalation or lymphatic transport, induce the same fibrogenic process, resulting in pleural fibrosis, or plaques if the scars are more circumscribed. These fibrogenic diseases are the result of a continuing process that is initiated upon deposition of the fibers and the consequent injuries that they induce.

Each time a population of fibers is deposited on the alveolar and pleural membranes additional injuries occur with consequent fibrogenesis. Depending upon the dose of fibers (concentration of fibers times the duration of exposure), the fibrogenic diseases can manifest clinically anywhere from 10 to 50 years after the initiation of exposure. Extremely heavy occupational exposures have resulted in even shorter latency periods before clinical signs and symptoms. Typically, the first symptom is shortness of breath upon exertion. This is caused by loss of lung elasticity as normal connective tissue components are replaced by stiffer, less compliant collagenous (scar) tissue. This individual typically is diagnosed with a “restrictive” lung disease, since he/she is restricted from taking a deep breath. In addition, as the scarring of asbestosis progresses, the normally thin alveolar-capillary membranes become thickened with collagen, thus, interfering with exchange of respiratory gases and contributing to the shortness of breath. Some individuals develop a “progressive” fibrogenesis that continues to worsen even after exposure to asbestos ceases. This is due to the cumulative effect of fibers which have been retained in the interstitial compartments of the lung following each asbestos exposure.

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LUNG CANCER:

Inhaled cigarette smoke damages the mucociliary escalator and reduces the clearance capacity of the lung. In chronic smokers there are likely to be areas of squamous metaplasia where the normal pseudostratified ciliated columnar epithelium is replaced by a more simple squamous (flattened) epithelium. There is evidence that a squamous airway epithelium is more phagocytic than the normal columnar type, and this results in the sequestration of increased numbers of inhaled asbestos fibers in the lungs of cigarette smokers.

All of the asbestos types are complete carcinogens, meaning that asbestos alone is capable of inducing the genetic errors that can lead to cancer. Cigarette smoke contains many such carcinogens. Cancer is the loss of control of cell growth caused by errors in the genes that control cell proliferation. These errors can be induced by direct interaction with carcinogenic minerals like asbestos which causes aneuploidy and other chromosomal abnormalities. Genetic errors also are known to be produced by oxygen radicals that are generated from all forms of asbestos and from components of cigarette smoke. Epidemiology has shown that asbestos-exposed individuals who smoke are more likely to develop a lung cancer than those who only smoke. Since cigarette smoke damages the airway epithelium (as described above), increased numbers of fibers are retained in the airway walls of smokers, and asbestos fibers can bind carcinogens in cigarette smoke, the known carcinogenic effects of both agents can be amplified in a synergistic manner. It takes multiple genetic errors to finally cause an airway cell to develop into a cancerous clone. Asbestos exposures, day after day, can cause errors in genes that control normal cell proliferation. Latency periods of 20-60 years result as the genetic damage to genes such as k-ras and p53 is repaired, as cells with genetic errors are programmed to die, and as the immune system continues to battle the aberrant cells. In those individuals who are susceptible to developing a cancer, it is obvious that these formidable defense mechanisms have not been sufficient, and at least one cell with a series of genetic errors has escaped to produce the tumor.

MESOTHELIOMA:

Cigarette smoking has no apparent influence on the development of malignant mesothelioma. The only established environmental cause of this cancer in North America is exposure to asbestos. All of the asbestos varieties cause mesothelioma through induction of genetic errors by binding DNA and/or generating oxygen radicals (as described above) in the mesothelial cells that line the pleural or peritoneal cavities. On a fiber per fiber basis, the amphibole asbestos varieties appear to be more potent than chrysotile as causative agents of mesothelioma; however, chrysotile breaks down to smaller fibers that are more easily transported through the lymphatic vessels to the pleural tissues where the "target cells" for mesothelioma reside. In some studies at autopsy, investigators have found a preferential accumulation of chrysotile fibers in pleural and mesotheliomatous tissues. Mesothelioma is caused by cumulative exposure and because of this, it is impossible to exclude a particular exposure as contributing to or

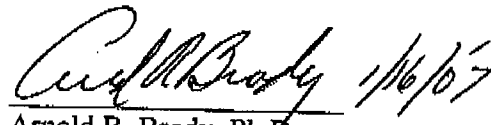
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causing the disease. As with lung cancer, mesothelial tumors typically are derived by clonal expansion of a single transformed cell. Also, similar to lung cancer, the latencies before clinical manifestation of mesothelioma are 10 to 50 years or more. Even though mesothelioma is a dose-responsive disease, this tumor has been shown to develop in susceptible individuals with brief exposures, and no safe or threshold level of exposure to asbestos has been determined for mesothelioma. There is no effective treatment for this deadly tumor.

The sequence and details of the disease process described above were established through a long series of correlative studies using experimental animals and human tissues. Humans and animals exposed to asbestos fibers develop the same diseases at the same anatomic sites. Inasmuch as it is impossible to follow the precise temporal events in the lungs of humans, animals (typically rats or mice) exposed to aerosols of asbestos fibers are used to elucidate the intricate details of the disease process. In addition, all of the cell types described have been removed from humans and animals and studied in vitro to elucidate cellular and molecular details that cannot be studied in the living organism.

I have testified previously in asbestos property damage cases on behalf of building owners.

The basis for these opinions are my education and over 30 years of training and experience with asbestos-induced diseases, including the almost 200 peer-reviewed and invited articles listed in my CV (attached) and the references therein.

My current fee for litigation activities is \$400 per hour.


Arnold R. Brody, Ph.D.
Professor

CURRICULUM VITAE

Name: Arnold R. Brody, Ph.D.

Address: Department of Molecular Biomedical Sciences
North Carolina State University
4700 Hillsborough St.
Raleigh, NC 27606

Date and Place of Birth: March 24, 1943, Boston, MA

Citizenship: United States

Marital Status: Married, 2 children

Education:

June 1961 Nashua Public High School, Nashua, NH
June 1965 B.S. Colorado State University, Zoology
June 1967 M.S. University of Illinois, Functional Vertebrate Anatomy
June 1969 Ph.D. Colorado State University, Cell Biology Ultrastructural Cytology

Brief Chronology of Employment:

1969-1972 Post-Doctoral Fellow (NIH) with the Acarology Laboratory, Ohio State University, Columbus, OH
1972-1978 Assistant Professor, Department of Pathology, University of Vermont, Burlington with the Vermont Specialized Center of Research in Pulmonary Disease
1978-1984 Senior Staff Fellow, Laboratory of Pulmonary Function and Toxicology, National Institute of Environmental Health Sciences
1984-1987 Research Biologist (Tenured), Laboratory of Pulmonary Pathobiology, National Institute of Environmental Health Sciences
1987-1990 GS-14
1978-1993 Head, Pulmonary Pathology Group, Laboratory of Pulmonary Pathobiology, National Institute of Environmental Health Sciences
1978-1993 Adjunct Professor, Department of Pathology, and member of Graduate School Faculty, Duke University, Durham, NC
1985-1993 Faculty: Curriculum in Toxicology, University of North Carolina College of Medicine, Chapel Hill, NC
1991-1993 GS-15 (Full Professor with Tenure)-NIEHS
1993 -2006 Full Professor, Department of Pathology, and Department of Environmental Health Sciences, Tulane University Medical Center, New Orleans, LA
1993 -2006 Director, Lung Biology Program, Center for Bioenvironmental Research, Tulane University Medical Center, New Orleans, LA
1993 -2006 Graduate Faculty, Molecular and Cellular Biology Program, Tulane University Medical Center, New Orleans, LA
1999 -2006 Vice Chairman, Department of Pathology, Tulane University Medical Center, New Orleans, LA
2006- Date Professor, Department of Molecular Biomedical Sciences, North Carolina State University, Raleigh, NC

Military Service:

1961-1963 Army ROTC

Honors and Invited Participation:

- 1967 Outstanding teaching assistant, Department of Zoology, University of Illinois.
- 1969 Sigma Xi, Biological Honorary.
- 1974 Visiting Scientist: Pneumoconiosis Research Center, Cardiff, Wales.
- 1977 Research Fellowship: Institut National de la Sante et de la Recherche Medicale, Paris, France.
- 1978 Invited Participant and Oral Presentation: Aspen Lung Conference on Immunology of the Lung.
- 1979 Chairman: Use of SEM and associated techniques (DES) in studies of pulmonary pathobiology at Annual Electron Microscopy Society Mtgs.
Invited participant: IARC Mtg. on Biological Effects of Mineral Fibers, Lyon.
- 1980 Invited Presentation: Aspen Lung Conference on Environment and the Lung.
- 1981 Chairman: Use of electron microscopy and associated techniques in studies of pulmonary structure, function and disease at Annual Electron Microscopy Society Meetings.
- 1982 Invited Participant: Aspen Lung Conference on Pulmonary Secretions and Fluids.
Invited Presentation: International Conference on Occupational Lung Disease, Chicago, IL.
Invited Presentation: 4th Annual Occupational and Environmental Health Conference, Salt Lake City, UT.
Invited Presentation: Conference on The In Vitro Effects of Mineral Dusts, Little Rock, AR.
Invited Participant: NIH Workshop on the Fibroblast in Interstitial Lung Disease.
- 1983 Invited Participant and Elected Member of Committee on Basic Science: EPA Task Force on Environmental Lung Disease, Washington, DC, March.
Invited Tutorial on Pathogenesis of Asbestos-Induced Lung Disease: Annual Electron Microscopy Soc., Detroit, April.
Invited presentation: Aspen Lung Conference on Pathobiology of Pulmonary Emphysema, June.
Invited Paper: Theodore F. Hatch Symposium on Occupational Health, U. of Pittsburgh, September.
- 1984 Chairman: Symposium on Mechanisms of Acute Lung Injury. FASEB, St. Louis, MO.
Member NIH Study Section: Isolation and Characterization of Human Lung Cells.

Invited Speaker: Rochester International Conference on Environmental Toxicity, Rochester, NY, June.
 Faculty: Pulmonary Pathology Course, University of Vermont, Department of Pathology, August.
 Scientific Advisory Committee and Invited Presentation: In Vitro Effects of Mineral Dusts, Schluchsee, FRG, October.

- 1985 Visiting Professor: Department of Pathology, Instituto Nacional DeCardiologia, Mexico City, January.
 Visiting Professor: Department of Biology, University of California, Santa Barbara, April.
 Chairman: Symposium on Pulmonary Response Following Particulate Exposure in Experimental Animals, Annual Meeting American Thoracic Society, Anaheim, CA, May.
 Visiting Professor: Department of Pathology, State University of New York at Stony Brook, June.
 State-of-the-Art Presentation: Cellular Pathobiology of Asbestosis at 28th Annual Aspen Lung Conference, June.
 Awarded: John P. Wyatt Traveling Fellowship In Environmental Pathology.
 Invited Speaker: Depts. of Pathology and Medicine Pulmonary Seminar Program, Washington University, St. Louis, September.
 Standing Member of Merit Review Board: Respiratory Disease Section, Veterans Administration, Washington, DC, September 1985-88.
- 1986 Invited Speaker: Pathology Grand Rounds, Harvard Medical School, Boston, January.
 Invited Speaker: Conference on Silicosis and Mixed-Dust Pneumoconiosis Chantilly, France, April.
 Visiting Scientist: Institut Nationale de Sante et Recherché Medicale, Paris, France, May - June.
 Invited Speaker: Gordon Conference on Pulmonary Biology, New London, New Hampshire, July.
 Invited Speaker: Electron Microscopy Society of America, Albuquerque, August.
 Invited Speaker: Third International Conference on Environmental Lung Disease, Montreal, October.
- 1987 Appointed: National Academy of Sciences Panel on Pulmonary Toxicology.
 Outstanding Performance Cash Award from NIH, March.
 Chairman: Symposium on Cellular Mechanisms of Occupational Lung Injury. Annual Meeting American Thoracic Society, New Orleans, May.
 NIH Study Section: Ad Hoc Panel to review RFA on subpopulations of pulmonary interstitial cells.
 Invited Speaker: IARC Conference on Mineral Fibres in the Non-Occupational Environment, Lyon, France, September.
 Keynote Address: "Mechanisms of Particle-Induced Lung Injury" American College of Chest Physicians, Atlanta, October.
 Invited Speaker: Lung Dosimetry: Extrapolation Modeling of Inhaled Particles and Gases, Duke University, October.
- 1988 Appointed: Visiting Committee: Harvard School of Public Health, Respiratory Biology Program, March.

Outstanding Performance Cash Award from NIH, March.

Visiting Scientist: University of Rochester School of Medicine, Environmental Health Sciences Center, March.

Organizer and Faculty: Course on Environmental Lung Disease, Mexico City, March.

Invited Speaker: Biological Interactions of Inhaled Mineral Fibers and Cigarette Smoke, Seattle, April.

Chairman: Symposium on "Pulmonary Macrophage Biology and Interstitial Lung Disease" Annual FASEB Conference, May.

Chairman and Invited Speaker: Symposium on "Biochemical and Molecular Mechanisms of Alveolar Fibrosis," Annual Meeting American Thoracic Society, May.

Invited Speaker: VIIIth International Pneumoconioses Conference, Pittsburgh, August.

Scientific Advisor: Workshop on In Vitro Effects of Mineral Dusts, Sherbrooke, Canada, September.

Chairman: "Mechanisms of Cellular Response to Inhale Substances," IIIrd Chicago Conference on Occupational Lung Disease, October.

Science Advisory Board: Health Effects Institute, Boston.

1989

Invited Speaker: Congressional Subcommittee on Environment and Technology. Washington, DC, January.

Invited Speaker: Dept. of Environmental Hygiene, University of Gothenburg, Sweden, March.

Invited Speaker: 3rd Alexis Carrell Conference on Accelerated Atherosclerosis, Washington, DC, March.

Invited Speaker: 2nd GERP Conference on Occupational Lung Disease, Paris, France, March.

Invited Speaker: Dept. of Pathology, Baylor College of Medicine. Houston, TX, May.

Conference Organizer and Speaker: Fiber Toxicology, Research Triangle Park, NC, June.

International Advisory Committee: Appointed as standing member for The Annual Aspen Lung Conference.

Invited Participant: XIVth Annual European Symposium on Hormones and Cell Regulation, Mont St-Odile, France, September.

1990

Invited Speaker: Symposium on Molecular Biomarkers of Disease, NIEHS, February.

Invited Participant: Banbury Center Conference on Molecular Mechanisms of Fiber Cytotoxicity and Carcinogenesis, Cold Spring Harbor, NY, March.

Program Committee and Session Chair: Mechanisms of Particle-Overload Induced Lung Disease, Rochester, May.

Session Chair: Symposium on Cell-Cell Interactions in the Lung, World Conference on Lung Health, Boston, May.

State-of-the-Art Speaker: Aspen Lung Conference on Mechanisms of Lung Repair, Aspen, CO, June.

Program Committee and Invited Speaker: The Third Wave of Asbestos Disease, Exposure to Asbestos in Place, NY, June.

Invited Participant: International Congress on Inflammation, Barcelona, June.

Invited Participant: Alveolar Macrophages in the Clearance of Inhaled Particles, Oxford, September.

Program Committee and Speaker: Biannual Symposium on Pulmonary Fibrosis, Stowe, VT, October.

1991

NIH Study Section: Specialized Centers of Research in Pulmonary Disease, Bethesda, January.

Visiting Scientist: Environmental Science Center, Univ. of Calif., Davis, February.

Visiting Pulmonary Scholar: Univ. of Wisconsin School of Medicine, March.

Keynote Speaker: American Association of Respiratory Therapy, Providence, March.

Session Chair and Speaker: Mechanisms of Pulmonary Fibrosis Amer. Assoc. of Pathologists Minisymposium, Atlanta, April.

Invited Speaker and Member of the Environmental Lung Disease Working Group of the Pulmonary Diseases Advisory Council, NHLBI Workshop, Washington, DC, May.

Christie Memorial Lecturer: Australian Society for Experimental Pathology, Adelaide, Australia, September.

Chairman: Basic Science Symposium, Fourth International Conference on Environmental Lung Disease, Montreal, September.

Chairman: Biological Reaction to Dust, Seventh International Symposium on Inhaled Particles, Edinburgh, September.

Invited Speaker: Woods Hole Conference on Pulmonary Biology, October.

1992

Invited Speaker and Organizer: Environmental Pulmonary Disease, Cuernavaca, Mexico, January.

Invited Faculty: Scientific Frontiers of Occupational Pulmonary Medicine, Miami, May.

Scientific Committee: Workshop on Durability of Inhalable Minerals, Lyon, France, September.

Invited Speaker: International Conference on Pulmonary Vascular Remodeling in Health and Disease, London, September.

Scientific Committee and Speaker: Seventh International Pulmonary Fibrosis Colloquium, Cambridge, U.K., October.

Visiting Professor: University of Michigan School of Medicine, October.

Invited Speaker: Cochin Hospital, Paris, November.

1993

Member: American Thoracic Society Task Force Developing a Strategic Plan for Lung Research into the 21st Century.

Invited Speaker: FASEB Conference on Pulmonary Pathobiology, New Orleans, April.

Invited Participant: Workshop on Interactions of Particles with the Lung, ATS Meeting, San Francisco, May.

Invited Speaker: 4th European Meeting of Environmental Hygiene, Wageningen, The Netherlands, June.

Invited Speaker and Organizing Committee: Cytokines and Lung Inflammation, Institut Pasteur, Paris, June.

Invited Speaker: 24th International Conference in Occupational Health, Nice, September.

Invited Speaker: Effects of Mineral Dusts on Cells, Paris, October.

Member: Scientific Site Visit Committee to the Institut Pasteur, Paris,

October.

Invited Speaker: World Congress on Inflammation, Vienna, October.

1994

Invited Participant: Colloquium on Particulate Air Pollution and Human Mortality and Morbidity, National Academy of Sciences Conference, Irvine, CA, January.

Invited Speaker: European Society for Clinical Investigation, Toledo, Spain, April.

Invited Speaker: American Lung Association, Science Day for the Media, New York, September.

Scientific Co-Chairman and Member of Organizing Committee: Eighth International Conference on Pulmonary Fibrosis, Dijon, France, October.

Ad Hoc Participant: Lung Biology Study Section, Bethesda, October.

Invited Speaker: Institute of Preventive and Clinical Medicine, Bratislava, Slovakia, November.

1995

Invited Speaker: 5th International Inhalation Symposium, Hannover, Germany, February.

Invited Speaker: American College of Chest Physicians, 5Th International Conference on Environmental and Occupational Lung Disease, Orlando, March.

Invited Speaker: NIEHS-Sponsored Public Health Symposium, Mexico City, March.

Chairman and Invited Speaker: Environmental Biology '95, Minisymposium on Environmental Pathology and Toxicology, Atlanta, April.

Invited Speaker: American Thoracic Society Annual International Conference, Symposium on Interstitial Pulmonary Fibrosis, Seattle, May.

Co-Chair and State-of-the-Art Speaker: 38th Annual Aspen Lung Conference, Environmental Lung Disease, Aspen, June.

Chairman: Session on Fiber Dissolution, British Association for Lung Research, Edinburgh, September.

Selected: Wellcome Visiting Professor in the Basic Medical Sciences, sponsored by the Burroughs Wellcome Fund, November.

1996

NIH SCOR (Fibrotic Lung Diseases) Study Section, Washington, DC, January.

NIH Lung Biology Pathology Study Section, Washington, DC, February.

Featured Speaker: American Thoracic Society Annual Conference, Symposium on Cell Activation in Lung Injury, New Orleans, May.

Invited Speaker: NATO Advanced Study Institute, Vascular Endothelium, Crete, Greece, June.

Invited Speaker and Scientific Organizing Committee: International Conference on Toxicology of Natural and Man-Made Particles, Lake Placid, NY, September.

Invited Participant and Workgroup Member: Association of Occupational and Environmental Clinics, Workshop on Particulates and Chronic Airways Disease, Pittsburgh, October.

Invited Speaker: Annual Woods Hole Conference on Lung Biology, Woods Hole, MA, October.

Invited Speaker and Organizer: Ninth International Colloquium on Pulmonary Fibrosis, Oaxaca, Mexico, November.

- 1997
- Invited Speaker: Dean's Distinguished Faculty Forum, Tulane University Medical Center, New Orleans, February.
 - Participant: NIH Lung Biology and Pathology Study Section, Bethesda, MD, February.
 - Invited Speaker and Co-Chair: Mediator and Signal Transduction in Lung Injury Models, Annual Meeting of the American Society for Investigative Pathology, New Orleans, April, 1997.
 - Invited Speaker and Co-Chair: Cellular Mechanisms of Pulmonary Fibrosis, Annual Meeting of the ATS, San Francisco, May.
 - Invited Speaker: World Health Organization Conference on Lung Injury, Corfu, June.
 - Participant: NIH Special Study Section--Oxidative Lung Injury, Research Triangle Park, NC, August.
 - Scientific Advisor and Participant: Second International Meeting on Oxygen/Nitrogen Radicals and Cellular Injury, Chapel Hill, NC, September.
 - Invited Speaker: International Conference on Occupational Health, Kyoto, Japan, October.
 - Visiting Professor: Tainan Medical College, Taiwan, October.
 - Visiting Professor: Beijing Medical College, Beijing, China, October.
 - Invited Speaker: University of California, Davis, College of Medicine, November.
- 1998
- Study Section: Member of MIT Site Visit Team for the Environmental Science Center, Boston, March.
 - Special Study Section: Children and Environmental Health, NIEHS, Research Triangle Park, NC, March.
 - Invited Speaker: EuroConference on Therapeutic Approaches for Diseases of the Respiratory Tract, Institut Pasteur, Paris, April.
 - Invited Speaker: Respiratory Biology at the American Society for Investigative Pathology, San Francisco, April.
 - Session Chairman: Mechanisms of Lung Injury, American Thoracic Society, Annual Conference, Chicago, April.
 - Invited Speaker and Organizer: Tenth International Colloquium on Pulmonary Fibrosis, Siena, Italy, October.
 - Session Chairman: Symposium on Systemic Sarcoidosis, National Heart, Lung, and Blood Institute Conference, New Haven, October.
 - Member: National Institute of Environmental Health Sciences, Environmental Health Sciences Review Committee, 1998-2002.
- 1999
- Member: ATS Committee on Research Advocacy, American Thoracic Society, 1999-2000.
 - Invited Speaker: 6th International Conference on Environmental and Occupational Lung Diseases, American College of Chest Physicians, Vancouver, February.
 - Member: ATS Government Relations Committee, American Thoracic Society, 1999-2000.
 - Invited Speaker: Signal Transduction and Growth Factors in Molecular Toxicology, National Institute of Environmental Health Sciences, Research Triangle Park, NC, March.
 - Member: Research Grants Review Committee, American Lung Association, 1999-2000.

Invited Speaker: Cytokines: Biology, Gene Regulation and Role in the Pathogenesis of Lung Disease, Society of Toxicology, New Orleans, March.

Invited Speaker: Experimental Biology, Recent Advances in Molecular Mechanisms and Pharmacological Interventions of Lung Fibrosis, Washington, D.C., April.

Invited Speaker: Tissue Repair and Fibrosis, 4th World Congress on Inflammation, Paris, June.

Keynote Speaker: Seventh Symposium on Particle Toxicology, Maastricht, The Netherlands, October.

2000

Invited Panelist and Speaker: Advanced Research Cooperation in Environmental Health, 7th Biennial Symposium on Minorities and Cancer, Washington, D.C., February.

Invited "Special Lecturer": 4th Conference on Acute Lung Injury, Tokyo, Japan, February.

Invited Speaker: Update in Occupational Lung Disease, Johns Hopkins University, Baltimore, March.

Invited Speaker: Andrews' 15th Annual Asbestos Conference, Andrews Publications, New Orleans, April.

Chairman and Speaker: ATS-NIEHS Workshop, Toronto, Canada, May.

Study Section: Cancer and Smoking Disease Program, Nebraska Department of Health and Human Services, Omaha, May.

Scientific Committee and Conference Summarizer: 11th International Colloquium on Lung Fibrosis, Stockholm, Sweden, September.

2001

Member: ATS Committee on Research Advocacy, American Thoracic Society, 2000-2001.

Sabbatical Leave, Duke University, Division of Pulmonary Medicine, January-April; Institut Pasteur, Paris, May-June.

Study Section: University of California, Tobacco-Related Disease Research Program - Pulmonary Disease Review Committee, San Francisco, March.

Invited Speaker: ATS 2001 Meet-the-Professor Seminar 97th International Conference, San Francisco, May.

Organizer and Speaker: Euroconference on Chronic Lung Diseases, Paris, June.

Study Section: NIEHS Environmental Health Sciences Review Committee Meeting, Research Triangle Park, NC, November.

2002

Special Study Section: National Heart, Lung, and Blood Institute Conference, Chevy Chase, MD, February.

Site Visit: NIEHS - National Jewish Medical and Research Center, February.

Study Section: University of California, Tobacco-Related Diseases Research Program - Pulmonary Disease Review Committee, San Francisco, April.

Site Visit: NIEHS Special Emphasis Panel - Harvard School of Public Health, Boston, May-June.

Study Section: American Heart Association, Lung, Resuscitation, and Respiration Study Group, Chicago, October.

Invited Speaker: International Conference on Lung Fibrosis, Geneva, October

Invited Speaker: 7th Congress of Asian Pacific Society of Respiriology, Taipei, November.

Invited Speaker: Liaocheng Peoples Hospital, Peoples Republic of China, November.

- 2003 Chairman – Study Section: National Heart, Lung, and Blood Institute Special Emphasis Panel, Molecular Targets and Interventions in Pulmonary Fibrosis, Columbia, MD, March.
 Study Section: University of California, Tobacco-Related Diseases Research Program – Pulmonary Disease Review Committee, San Francisco, March.
 Invited Lecture: Mechanism of TGF- β Activation by Reactive Oxygen Species Harvard School of Public Health, Boston, April.
 Invited Speaker: Andrews' 18th Annual Asbestos Litigation Conference, Andrews Publications, New Orleans, May.
 Site Visit: NIEHS - Harvard School of Public Health, Boston, June
 Invited Lecture: EPA Workshop on Mechanisms of Asbestos Fiber Toxicity and Carcinogenicity, Chicago, June.
 Study Section: NIH (LPBA) Lung Biology and Pathology, Washington, DC, June.
 Invited Speaker: XVII World Asthma Congress, St. Petersburg, Russia. July.
- 2004 Study Section: NIH (NHLBI) National Heart, Lung, Blood Institute, Special Review Committee for Program Project Grant/PO1, Lung Fibrogenesis and the Biology of Fibroblast, Rockville, MD, February.
 Study Section: NIH (NHLBI) National Heart, Lung, Blood Institute, Special Review Committee for Program Project Grant/PO1, Regulation of Respiratory Epithelial Cell Homeostasis, Rockville, MD, February.
 Chairman -Study Section: NIH NHLBI National Heart, Lung, Blood Institute, Special Emphasis Panel Granulomatous Lung Inflammation in Sarcoidosis, Rockville, MD., March.
 Study Section: University of California, Tobacco-Related Diseases Research Program – Pulmonary Disease Review Committee, San Francisco, April.
 Invited Speaker: St. Luke's Medical Center, Grand Rounds, "Asbestos-induced fibrogenesis: how unraveling of the molecular mechanisms will direct potential therapeutic approaches," Milwaukee, WI, September.
 Invited Speaker and Session Chair: 13th International Colloquium on Lung Fibrosis. Banff, Alberta, Canada, October.
- 2005 Invited Speaker: Shanghai International Respiratory Symposium. Shanghai, China, October.
 Invited Speaker: 8th International Meeting on Effects of Mineral Dusts and Nanoparticles. NIH Research Triangle Park, NC, October.
 Scientific Advisory Board: BioMarck Pharmaceuticals
- 2006 Invited speaker: University of Rochester College of Medicine, Department of Pulmonary Medicine, March
 Invited Speaker: 14th International Colloquium on Pulmonary Fibrosis, Frankfurt, Germany, September.
 Invited Speaker: International Mesothelioma Conference; Chicago, October
 Invited Chair: Amer Physiol Soc Conf; Physiological Genomics and Proteomics Of Lung Disease; Ft. Lauderdale, November

Committees:**NIEHS**

Chairman: GS-9/11 Promotions Committee (1988-91)

Standing Member: Animal Care Committee (1984-90)

Standing Member: Tenure Review and Promotions Committee (1991-93)

Tulane

Department of Pathology - Tenure and Promotions (1993 - Present)

- Delegate to the Basic Science Faculty

- Strategic Plan Co-Chair

President's Search Committee for Senior Vice President for the Health Sciences

President's Search Committee for Director of the Center for Bioenvironmental Research

Chairman: Subcommittee on Training Grants

Chairman: Dean's Pharmacology Department Review

Standing Member: Cancer Center Steering Committee

Dean's Pharmacology Chair Search Committee

Elected Member: Molecular and Cellular Biology Steering Committee

Chairman: Dean's Forum for Advances in Research Committee

Member: Dean's Personnel and Honors Committee (1999-Present)

Elected Member: Faculty Advisory Committee

Appointed: Center for Bioenvironmental Research Faculty Council

Editorial Board

American Journal of Pathology

American Journal of Physiology: Lung, Cellular, and Molecular Physiology

American Journal of Respiratory, Cell, and Molecular Biology

Current Respiratory Medicine Reviews

Journal of Environmental Pathology, Toxicology, and Oncology

Journal of Inflammopharmacology

Journal of Organ Dysfunction

Section Editor (Pathology)

Journal of Lipid Mediators and Cell Signalling

Review Manuscripts for:

American Journal of Respiratory and Critical Care Medicine

Chemico-Biological Interactions

Environmental Health Perspectives

Environmental Research

FASEB Journal

Journal of Cellular Physiology

Journal of Clinical Investigation

Journal of Immunology

Journal of the American Physiological Society

Laboratory Investigation

Science

Toxicology and Applied Pharmacology

American Journal of Pathology

Active Memberships:

AAAS - American Association for the Advancement of Science
 American Thoracic Society - Assembly Nominating Committee
 FASEB - Federation of American Societies for Experimental Biology
 Sigma Xi (Biological Honorary)

Peer-Reviewed Publications

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2. Brody, A. R., and Wharton, G. W. Dermatophagoides farinae: Ultrastructure of lateral opisthosomal dermal glands. Trans. Amer. Micros. Soc. 89(4):499, 1970.
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